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Genetic and Environmental Contributions to Stability in Loneliness Throughout Childhood

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Heritability estimates based on two small cross-sectional studies in children indicate that the genetic contribution to individual differences in loneliness is approximately 50%. A recent study estimated the genetic contribution to variation in loneliness in adults to be 48%. The current study aims to replicate and expand these findings by conducting longitudinal analyses in order to study causes of individual differences in stability of loneliness throughout childhood. Univariate and multivariate longitudinal analyses are conducted in a large sample of young Dutch twins. Information on loneliness comes from maternal ratings on the Child Behavior Checklist. Using an average score of loneliness over ages 7, 10, and 12, results from the two previous studies are replicated and a heritability estimate of 45% is found. The remaining variance is accounted for by shared environmental influences (12%), and nonshared environmental influences (43%). The longitudinal analyses, however, show that heritability is 58% at age 7, 56% at age 10, but drops to 26% at age 12. A parallel increase in influences of shared family environment is observed, explaining 6% of the variance at age 7, 8% at age 10 and 35% at age 12. The remaining variance is explained by relatively stable influences of nonshared environmental factors. Stability in loneliness is high, with phenotypic correlations in the range of 0.51–0.69. This phenotypic stability is mainly caused by genetic and nonshared environmental influences. The results indicate the importance of both innate as well as nonshared environmental factors for individual differences in loneliness. Further, different results between causes of individual differences for the average score of loneliness and results for age 12 from the longitudinal analyses, indicate the importance of longitudinal analyses with data at well-defined ages.

KEY WORDS: loneliness; childhood; heritability; longitudinal; stability

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INTRODUCTION

The British empiricist John Locke (1632–1704) suggested that humans come into the world a tabula rasa, born with no built-in mental content and defined by events after birth. Infants were thought to be born with no inherent talents or temperaments, shaped entirely by parenting, culture, and society. In contrast, Darwin's [1872] treatise on the expression of emotions in humans and animals made a strong case for the heritability of social dispositions and behaviors. Situational factors nevertheless continue to be the focus of emphasis in investigations of social cognition and behavior [e.g., Aronson, 2003; Gilbert et al., 1998; Harlow and Harlow, 1973].

Progress over the past two decades in behavioral genetics has called into question the emphasis on situational factors alone. Human infants are born to an extended period of dependency and cannot survive to contribute genes to the gene pool unless they elicit care and nurturance and their caregivers are motivated to provide this care and nurturance. In this context, loneliness has been conceived as an aversive response to a threat to connections with others which motivates the individual to re-establish or repair these connections [Cacioppo et al., 2006a]. The purpose of the present study is to investigate how genetic and environmental factors contribute to individual differences in loneliness in children.

Understanding the origins, nature and course of loneliness is important for clinical purposes as well as for scientific purposes. Most of the research on loneliness has concentrated on either college-age students or elderly persons and development or stability of loneliness is seldom taken into account. In these studies loneliness is associated with poorer mental, physical well-being [e.g., Perkins, 1991; Gupta and Korte, 1994; Cacioppo et al., 2003] and mortality [Penninx et al., 1997; Herlitz et al., 1998]. Loneliness, for instance, is strongly associated with poor emotional well-being and depression [Anderson and Arnoult, 1985; Prince et al., 1997; Nolen-Hoeksema and Ahrens, 2002; Cacioppo et al., 2006b], and has been identified as a risk factor for the common cold [Cohen et al., 1997], elevated blood pressure [Hawkley et al.,

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2006] and heart diseases [Orth-Gomer et al., 1988; Sorkin et al., 2002].

So, although biological pathways from loneliness to illness are suggested in college students and adults, attention to loneliness during childhood is less pronounced. The focus of studies on loneliness during childhood has been on (environmental) causes of loneliness (e.g., peer rejection, peer relations, social adaptation). Loneliness in childhood is associated with unpleasant emotions and perceptions of unfulfilled relational needs, such as lack of companionship, support and affection [Asher and Paquette, 2003]. Children who are rejected by peers and who do not have close friendships report higher levels of loneliness and social dissatisfaction than others [Bukowski and Hoza, 1989; Asher et al., 1990; Renshaw and Brown, 1993]. As in college-students and adults, loneliness during childhood is associated with a number of aspects of maladjustment such as depression, suicide, poor self-concept, psychosomatic problems and ADHD [Cassidy and Asher, 1992; Renshaw and Brown, 1993; Boivin et al., 1995].

Little attention has been paid to the question whether genetic factors might influence the development of individual differences in loneliness. Twin and adoption studies provide a powerful instrument to investigate this question. To date, only two investigations have addressed the etiology of feelings of loneliness [McGuire and Clifford, 2000; Boomsma et al., 2005]. McGuire and Clifford [2000] examined the heritability of loneliness in children using an adoption study design and a twin study design. In their first study, 60 biologically related pairs and 64 unrelated pairs in adoptive families in the Colorado Adoption Project completed an 8-item self-report loneliness scale [Asher's general loneliness questionnaire; Asher et al., 1984] when they were 9, 10, 11, and 12 years of age. An average score across the four time points was used in the univariate analyses, providing insight in the causes of individual differences in loneliness in childhood, but not providing insight into the development of loneliness throughout childhood. In a second study, 22 monozygotic (MZ) twin pairs, 40 dizygotic (DZ) twin pairs, and 80 full-sibling pairs 8–14 years of age completed Asher's 16 item loneliness scale to assess loneliness in relation to their schoolmates [Asher and Weeler, 1985]. Univariate analyses (corrected for age and sex) results revealed significant genetic ($h^2 = 55\%$ and 48%, respectively, in Studies 1 and 2) and nonshared environmental contributions to individual differences in loneliness.

To examine the genetic and environmental determinants of loneliness in adults, Boomsma et al. [2005] conducted a large scale study on genetic and environmental contributions to loneliness based on data of 8,387 young adult and adult Dutch twins. They estimated the genetic contribution to variation in loneliness to be 48%, comparable to the study by McGuire and Clifford [2000]. Moreover, shared environmental factors were found to not contribute to resemblances between twin relatives, but instead unshared environmental factors accounted for the remaining variation in loneliness. Finally, Boomsma et al. found no evidence for gender or age differences in genetic architecture.

These studies provide important evidence for the significance of genetic factors in explaining individual differences in loneliness at different ages but the work by McGuire and Clifford [2000] remains the only, however relatively small, genetic study of loneliness in children. The current research was designed to add to this work. First, we utilized a large sample size to better distinguish between genetic and environmental sources of variation, especially genetic and shared environmental factors. Second, our sample consisted of large age-restricted samples with measures strictly at age 7, 10, and 12. Finally, we investigated the stability in loneliness in children from age 7 to 12 using a longitudinal design. The longitudinal design is important and an improvement for two reasons. First, it allowed us to examine whether loneliness in children is a temporary state or a continuous trait. Second, the longitudinal design allowed us to determine the genetic architecture of loneliness at specific ages throughout childhood. For example in studies on the influences of shared environment in IQ, genetic influences diminish after the age of 12 [Bartels et al., 2002] and in a study on causes of individual differences in exercise behavior, shared environment is important in explaining individual differences in adolescent, but individual differences in exercise behavior in adults is accounted for by genetic factors [Stubbe et al., 2005]. Finally, shared environmental influences tend to increase for Internalizing Behavior throughout childhood [Bartels et al., 2007].

In summary, the aim of the current study is to replicate the findings of the two previous studies and expand these findings by conducting longitudinal analyses in order to study causes of individual differences in stability of loneliness throughout childhood. Information on loneliness at ages 7, 10, and 12 years is gathered based on summing of maternal ratings of two items of the Child Behavior Checklist [CBCL; Achenbach, 1991]. Univariate genetic analyses on an average score across ages are used for the replication study. A multivariate genetic design is used to estimate influences of genetic and environmental factors on loneliness at the distinct ages. More importantly, this longitudinal design is used to gain insight into genetic and environmental influences on the covariance between loneliness at the distinct ages.

MATERIALS AND METHODS

Participants

The data of the present study are derived from a large ongoing longitudinal study, which examines the genetic and environmental influences on the development of problem behavior in families with 3- to 12-year-old twins. The families are volunteer members of the Netherlands Twin Register, kept by the Department of Biological Psychology at the Vrije Universiteit in Amsterdam, the Netherlands [Boomsma et al., 2006; Bartels et al., 2007]. From 1986 onwards families with twins are recruited a few months after birth. Currently, 40–50% of all multiple births are registered by the Netherlands Twin Registry. Questionnaires were mailed to families within 3 months of the twins' seventh, tenth, and twelfth birthday. After 2–3 months reminders were sent and 4 months after the initial mailing persistent non-responders were contacted by phone if financial resources were available [for details see Bartels et al., 2007]. Families whose addresses were unknown were included in the nonresponse group. Maternal ratings on loneliness were available for 7995 twin pairs (1329 MZM, 1336 DZM, 1535 MZF, 1248 DZF, 1308 DOSMF, 1239 DOSMF). Data analyses were conducted on raw data, so also data from twins with missing datapoint were included. The twins are from cohorts 1986–1991. No significant differences ($P > 0.05$) in loneliness are observed for twin pairs who participated at all ages versus twin pairs that dropped out of the study at one or more ages.

For 507 same sex twin pairs zygosity was based on blood group or DNA polymorphisms. For the remaining twins zygosity was determined by questionnaire items, filled in by the mother, about physical similarity and frequency of confusion of the twins by family and strangers [Goldsmith, 1991]. The classification of zygosity was based on a discriminant analysis, relating the questionnaire items to zygosity based on blood/DNA typing in a group of same-sex twin pairs. The zygosity was correctly classified by questionnaire in 93% of the cases [Rietveld et al., 2000].

Socioeconomic Status (SES) was obtained from a full description of the occupation of the parents when the children were 3 years of age. The level of occupation was coded according to the system used by Statistics Netherlands [CBS, 2003]. The code was based on the mental complexity of the work and ranged from low skilled to scientific work. An earlier comparison of the parental SES distribution with those obtained for the general Dutch population showed a slightly higher frequency of the middle and higher SES-groups [for details see Rietveld et al., 2004].

Measures

Two items from the CBCL were used (item 12 "Complains of loneliness" and 33 "Feels or complains that no one loves him/her"). Item selection was based in factor analyses on 6 items of the YASR [Boomsma et al., 2005]. The CBCL [Achenbach, 1991] is highly overlapping with the YASR, stems from the same taxonomy and is based on the same psychometric properties. The two items are correlated (polychoric correlations are 0.57 at age 7, 0.71 at age 10, and 0.68 at age 12) indicating that they measure the same construct and that addition of the two items gives a more complete picture of loneliness.

The CBCL is developed for parents to score the behavioral and emotional problems of their 4- to 18-year-old children and consists of 120 problem items that are scored by parents on a 3-point scale based on the occurrence of the behavior during the preceding 6 months: (0) if the problem item was not true of the child, (1) if the item was somewhat or sometimes true, and (2) if it was very true or often true. By adding the scores on the two items a score in the range of 0–4 could be computed at each age. For the replication study loneliness scores were averaged over occasions and recoded into the nearest (0 through 4) category. Subjects had to have at least scores on 2 items to be included in the replication study. Because of the low endorsement of highest category, this category was pooled with the previous one. The resulting loneliness variable thus had 4 categories. For the longitudinal study data at each age were recoded into two categories; (0) absence of loneliness; (1) presence of mild to severe loneliness, which was formally represented by categories 1, 2, 3, and 4. The dichotomous scores at ages 7, 10, and 12 were used in the longitudinal analyses. This restriction in categories was necessary in order to overcome multiple empty cells due to longitudinal data collection.

Data Analyses

Both the univariate and the longitudinal analyses are conducted in Mplus [Muthén and Muthén, 1998–2005]. The models used in our analyses are based on the classical twin design (see Fig. 1). In this design the relative contributions of genetic and environmental factors to individual differences in loneliness can be inferred from the different levels of genetic relatedness between MZ and DZ twins. Individual differences may be due to additive genetic (A), shared environmental (C) or nonshared environmental (E) factors [Neale and Cardon, 1992]. Additive genetic factors are correlated 1.0 in MZ twins, since MZ twins are genetically identical. For DZ twins, the additive genetic factors are correlated 0.5, because DZ twins share on average half of their segregating genes. The environment shared by a twin pair is assumed not to depend on zygosity, and thus shared environmental factors correlate 1.0 in both MZ and DZ twins. E or nonshared environment is by definition uncorrelated. All uncorrelated error is also absorbed in the E term.

Twin correlations (both for the average score and the age-specific score), phenotypic polychoric correlations (indicating

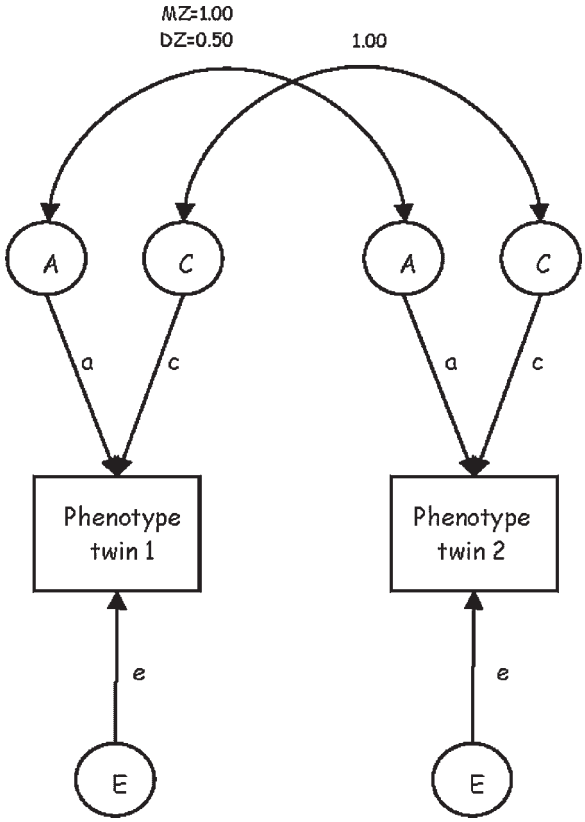

Fig. 1. The univariate twin model.

stability and change of loneliness over time), and cross-twin-cross-age polychoric correlations (e.g., loneliness for the oldest of the twin pair at age 7 and the youngest of the twin pair at age 12) for the monozygotic and dizygotic twins have been calculated. Tests for sex-differences in correlations are conducted by equating these to be equal for boys and girls. The twin correlations of the average score provide a first indication of the underlying sources of individual differences in loneliness. The phenotypic polychoric correlations give information on the longitudinal stability of loneliness throughout childhood. The age-specific twin correlations provide information on the causes of individual differences at ages 7, 10, and 12. The cross-twin-cross-age correlations give a first impression of the genetic and environmental contributions to stability and change of loneliness over time. If MZ cross-twin-cross-age correlations are higher than DZ cross-twin-cross-age correlations a first sign of genetic influences on stability of loneliness is gained. If MZ cross-twin-cross-age correlations are less than twice as high as the DZ cross-twin-cross-age correlation significant influences of shared environmental influences on the covariance is to be expected.

The Replication Study

In order to replicate the two previous behavior genetic studies on loneliness univariate genetic model fitting techniques in Mplus [Muthén and Muthén, 1998–2005] were used to obtain estimates of the genetic and environmental influences on variances of the average score of loneliness across childhood. The categorical trait loneliness was modeled to have an underlying continuous distribution with 3 thresholds (category 0, 1, 2, and 3). This underlying distribution has been termed the liability or vulnerability [Falconer and Mackay,

1996]. The continuous variation in liability may be both genetic and environmental in origin. The variance of the liability distribution was standardized. Influences of genetic, shared, and nonshared environmental factors on loneliness are estimated. Sex-differences in the magnitude of A, C, and E were tested by constraining correlations for boys and girls to be equal. Next, significance of the shared environmental effects was tested by fitting a AE model to the data. Model comparison was conducted by mean-adjusted robust Chi-square difference testing [Muthén and Muthén, 1998–2005].

The Longitudinal Analyses

To estimate heritability of loneliness at ages 7, 10, and 12 separately and to investigate the underlying sources of the stability of loneliness throughout childhood longitudinal data were used. Multivariate genetic model fitting techniques were used to obtain estimates of the genetic and environmental influences on variances and covariances. In this multivariate method not only the expectation for the within-pair covariances is taken into account but also the cross-trait as well as the within-person information. Further, increase in power is achieved through multivariate analyses, for instance by a repeated measurement design as used in this study. Provided that those repeated measurements correlate significantly with each other, this yields large gains in power [Schmitz et al., 1998].

In order to estimate influences of genetic, shared, and nonshared environmental factors on variance and covariance of loneliness throughout childhood a saturated model, also know as a Cholesky Decomposition or triangular decomposition was used (See Fig. 2). The Cholesky decomposition is descriptive and not driven by a specific developmental hypothesis. It decomposes a covariance matrix into genetic and non-genetic covariance matrices and is a first approach to obtain genetic and environmental correlations across time in longitudinal datasets.

RESULTS

Prevalence of loneliness at ages 7, 10, and 12 for boys is 14%, 18%, and 15% respectively. For girls prevalences are 17%, 20%, and 18%.

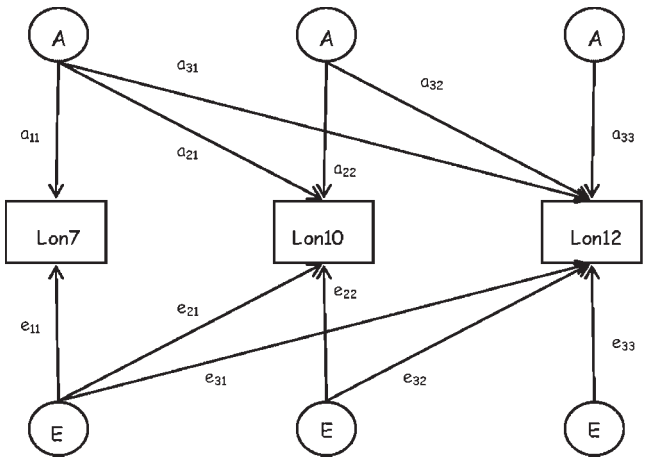

Fig. 2. The Cholesky Decomposition (for sake of simplicity the path diagram only contains the additive genetic and nonshared environmental influences. Shared environmental influences could be expressed in the exact same manner).

The Replication Study

No significant sex-differences were found for the twin correlations ($\chi^2 = 2.52$, df = 4, $P = 0.64$). MZ twin correlation for the average score of loneliness is 0.57 and DZ twin correlation is 0.35. The differences between MZ and DZ twin correlations indicate the significance of genetic influences. Furthermore, based on this pattern of twin correlations effects of shared environmental factors are to be expected.

The best fitting model for the average score of loneliness is an ACE model, since he dropping the shared environmental component from the model, gave a significant deterioration of fit ($P < 0.05$). Individual differences in loneliness (averaged over ages 7, 10, and 12) is for 45% accounted for by genetic factors. The remaining variance is accounted for by shared environmental factors (12%) and nonshared environmental factors (43%).

The Longitudinal Analyses

Phenotypic polychoric correlations over time are presented in the upper part of Table I. Stability in loneliness throughout childhood is indicated by the moderate to high correlations over time. A slight expected decrease in correlations is observed when time intervals increase (e.g. for $r_{10-12} = 0.69$ and $DZMr_{7-12} = 0.51$). However, the correlation between loneliness at age 7 and loneliness at age 12 is still highly significant, indicating loneliness to be stable over time.

Age-specific twin correlations derived from the longitudinal saturated model are given bold faced in Table I. No sex-differences in the correlations are found ($\chi^2 = 21.340$, df = 17, $P = 0.2115$). The differences between MZ and DZ correlations decrease over age, indicating a possible decrease in genetic influences and a parallel increase in shared environmental influences on loneliness. Cross-twin-cross-age correlations for MZ and DZ twins separately (Table I, *in italics*), give a first impression of the underlying sources of stability in loneliness over time. In general MZ cross-twin-cross-age correlations are larger than the DZ cross-twin-cross-age correlations, indicating influences of additive genetic factors on stability. The MZ correlations, though, are less than twice the DZ correlations, indicating influences of shared environmental factors on stability of loneliness throughout childhood as well. Moderate to large nonshared environmental influences on stability are expected based on the relatively low MZ cross correlations.

Next, to gain insight into the underlying sources of loneness throughout childhood, multivariate genetic analyses are conducted. Results of the longitudinal liability model estimations are given in Table II. Standardized estimated of A, C, and E for loneliness at ages 7, 10, and 12 are presented on the diagonal (Bold faced). Heritability of loneliness drops from 58% at age 7–26% at ages 12. An increase of shared environmental influences is observed (6% at age 7–35% at age 12).

Influences of A, C, and E on the covariances (indicating stability of loneliness over time) are represented by the off-diagonal elements.

Stability in loneliness from ages 7 to 12 is explained by additive genetic, shared environmental and nonshared environmental influences. Half of the stability, on average, is accounted for by additive genetic factors (56% between age 7 and 10, 45% between age 7 and 12, and 50% between age 10 and 12). An increase of the influences of shared environmental factors is also observed for the covariances. On average 15% of the stability is accounted for by shared environmental influences (5% between age 7 and 10, 18% between age 7 and 12, and 24% between age 10 and 12). The remaining covariance between loneliness at the distinct ages is accounted for by nonshared environmental influences (39% between age 7 and

TABLE I. Phenotypic Polychoric Correlations, Polychoric Twin Correlations (bold faced) and Polychoric Cross-Twin-Cross-Age Correlations (in italics) for MZ and DZ Twins

	Lon 7	Lon 10	Lon 12
Phenotypic			
Lon 7	1		
Lon 10	0.57	1	
Lon 12	0.51	0.69	1
	Lon 7 oldest[a]	Lon 10 oldest	Lon 12 oldest
MZ			
Lon 7 youngest[b]	**0.65**		
Lon 10 youngest	*0.35*	**0.66**	
Lon 12 youngest	*0.31*	*0.37*	**0.58**
DZ			
Lon 7 youngest	**0.33**		
Lon 10 youngest	*0.20*	**0.39**	
Lon 12 youngest	*0.25*	*0.29*	**0.49**

[a]"Oldest" refers to the first born of a twin pair.
[b]"Youngest" refers to second born of a twin pair.

10, 35% between age 7 and 12, and 27% between age 10 and 12), which include idiosyncratic experiences as well as measurement error.

DISCUSSION

The purpose of the current study was to investigate causes of individual differences in loneliness in children. About 15–20% of the children under study showed signs of loneliness at a certain age. Heritability of loneliness averaged across age is 45%. This is in line with the study by McGuire and Clifford [2000], who examined the heritability of loneliness in children using an adoption study design and a twin study design. They found that 50% is the variance in loneliness is accounted for by genetic factors. Our finding also replicates the finding by Boomsma et al., in a large population sample of adult twins.

However, results from the longitudinal analyses reveal an interesting picture on the genetic architecture of loneliness throughout childhood. Individual differences in loneliness at the distinct ages (7, 10, and 12) are accounted for by genetic as well and shared and nonshared environmental influences. A drop in genetic influences and a rise in shared environmental influences, though, is observed for loneliness at age 12. The two previous behavioral genetic studies of loneliness [Boomsma et al., 2005; McGuire and Clifford, 2000] do not find significant shared environmental influences. Two possible reasons for the discrepancy between findings for shared environmental findings between the current study and the study by McGuire et al., are (1) the use of an average score over ages and (2) lack of power. McGuire and Clifford use an average score over ages 9–12 in their adoption sample and 8–14 in their twin sample. The use of the average score could have masked a real change in genetic architecture over ages. Furthermore, lack of finding of shared environmental influences in the study by McGuire et al. could be due to power. They were not able to distinguish between genetic factors and shared environmental factors as the main source of familial aggregation in the small CAP sample. In general, a lack of power in twin studies, often due to small sample sizes, results in an overestimation of genetic influences and an underestimation of shared environmental influences [Martin et al., 1978; Neale and Cardon, 1992]. Discrepancy between the current study and the study by Boomsma et al. is that the study by Boomsma et al. was conducted in a different age group, that is, young adult and adult twins. The absence of shared environmental factors in adults could reflect the possible absence of family influences due to the fact that the twins no longer share their family environment.

The reason for the observed drop in heritability can only be guessed at, but a possibility might be that entering puberty causes changes in the strength of genetic and environmental influences. Puberty could influence the expression of genetic factors, but it also changes the landscape of our social environmental and interpersonal relationships in dramatic ways. It is this life transition that could lead children to become sensitive to new kinds of interpersonal relationships, situational factors, and social rejections—that is, to be subjected to all kinds of novel and powerful situational influences for awhile. We expect that as these children age and have had a chance to learn and adapt to these "new" biological/situational challenges, their heritable dispositions should begin to reemerge as major influences in loneliness, which is reflected in the result of the study with young adult and adult twins of the NTR [Boomsma et al., 2005]. We are currently collecting self-report data of the twins at ages 14, 16, and 18, so future studies might provide more evidence for the change in genetic and environmental influences on loneliness.

Another explanation for the presence of significant shared environmental influences in this study and the absence of shared environmental influences in adults and in the study by McGuire et al. is the measurement method. The data on loneliness during childhood in the current study are based on mother ratings, while in the other two studies data on loneliness are based on self-report. The use of an external rater (like mother report) could have influenced our results. Several studies, for example point out the presence of rater bias in studies which use a single external rater instead of self-report or multiple raters. Rater bias is confounded with shared environmental influences in a single rater design. Rater bias is estimated to account for about 10% of the individual differences in the phenotype under study and 10–15% of the phenotypic stability [Van der Valk et al., 2001, 2003; Bartels et al., 2003, 2004]. Sources of rater bias are stereotyping, employing different normative standards, or having certain response styles, that is, judging problem behaviors more or less severely. It is expected that rater bias in this sense will be a continuous process influencing the ratings at all ages, and in that way mimic stability in the trait under study. Less obvious, but not erasable is the fact these types of bias may change over time, for instance mothers change their opinion on certain kinds of behavior leading to change in rating style. Finally,

TABLE II. Standardized Estimates of Additive Genetic (A), Shared Environmental (C), and Nonshared Environmental (E) Influences on Variances and Covariances of Loneliness at Ages 7, 10, and 12 (bold faced)

	A			C			E		
	7	10	12	7	10	12	7	10	12
7	**0.58**			**0.06**			**0.36**		
10	0.56	**0.56**		0.05	**0.08**		0.39	**0.36**	
12	0.45	0.50	**0.26**	0.18	0.24	**0.35**	0.37	0.25	**0.39**

levels of parental psychopathology could affect ratings of problem behavior in their children. Several studies suggest that depression in mothers may lead to their overestimating their children's symptomology [Fergusson and Horwood, 1987]. Still, after taking this limitation of maternal ratings into account, our study is the first to show the effects of genetic and environmental influences on loneliness at distinct ages and the stability of it throughout childhood. If rater bias is present, the influences of shared environment on loneliness and it's stability found in this study, might be slightly overestimated [maximal 10% based on previous studies; Bartels et al., 2003, 2004], but our estimate of shared environmental influences at age 12 is to high to be explained be rater bias solely, providing evidence for the effects of "real" shared environmental factors.

The phenotypic polychoric correlations using the longitudinal data at ages 7, 10, and 12 revealed high temporal stability of individual differences in loneliness. The median (2-year) temporal stability in loneliness between the ages of 10 and 12 was 0.69, the median (3-year) temporal stability between the ages of 7 and 10 was 0.57, and the median (5-year) temporal stability between the ages of 7 and 12 was 0.51. We further found that the underlying sources for this stability in loneliness included genetic, shared and nonshared environmental influences. Stability, though, is mainly accounted for by genetic and nonshared environmental influences, while shared environmental influences are less important (15% of the covariance), but an increase in importance of shared environmental factors on stability is observed.

The different results between causes of individual differences for the average score of loneliness and results for age 12 from the longitudinal analyses, indicate the importance of longitudinal analyses with data at well-defined ages. Furthermore, the results of our study shed new light on the causes of individual differences in loneliness in children at specific ages and stability of loneliness throughout childhood and are important for clinical purposes.

Clinical Implications

The findings of our study are important for clinical as well as theoretical reasons. The longer an individual continues along a maladaptive pathway, the more difficult it is to reclaim a normal developmental trajectory [Scroufe, 1990]. Research on child development suggests that early recognition of problems and causes of stability foster development of successful intervention and prevention in families [Hermanns and Leu, 1998]. Accordingly, our finding that children, who are identified as feeling lonely at a young age, are good candidates to show chronic feelings of loneliness throughout childhood, suggests that active intervention programs may prove beneficial for these children. Moreover, the increasing importance of shared environmental influences suggests that intervention in the child's environment could help to prevent and or cure the loneliness trait in children. Thus, our data argue against a "wait and see" policy. An active strategy to help children who shows signs of loneliness is not only important to mitigate the possibility that these individuals will develop these feelings chronically, but it may also help avoid the long-term effects of loneliness. At a young age, loneliness might not cause direct harm, but indirect effects are to be expected. Loneliness, for instance, may impair sleep in children as it does in adults, and sleep debt has been related to lowering of glucose intolerance, increasing sympathetic tonus and diminishing cortisol regulation [Spiegel et al., 1999]. Further, loneliness has been found to predict depressive symptoms, even after controlling for the obvious depression related factors such as demographic characteristics, perceived stress and social support [Cacioppo et al., 2006b]. According to the WHO (www.who.int, 2004) depression is the leading cause of disability and the 4th leading contributor to the global burden of disease. Within several years, depression is projected to be the most important cause of disease burden. Reducing loneliness in children may be a first step toward lessening the burden of chronic depressive symptoms in adults.

Limitations

A limitation of the study is the use of only two items of the CBCL to indicate the level of loneliness. This limitation should be noticed when interpreting the results. From a developmental perspective the use of the same two items throughout childhood gives the possibility to investigate the underlying sources of stability of loneliness. However, one can imagine that the concept of loneliness changes over lifetime. For children aged 7 or 10, the items "Complains of loneliness" and "Feels or complains that no one loves him/her" can be rather straightforward. However besides the fact that feelings of loneliness can change while entering puberty (as mentioned above), the concept of loneliness can also become more complex during puberty. A two item construct might be a simplistic approach during this age period, possibly reflected in the drop of genetic factors. However, the rather stable estimate of nonshared environmental influences provides no indication for a rise in measurement error. We, however, are convinced that our limitations could also be considered to be an advantage. By using the two item construct based on CBCL data we were able to investigate the sources of individual differences in loneliness in a large longitudinal genetically related sample, making this an unique study in the world.

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